charge, free from pain, even though there may still continue to be a moderate amount of ethmoidal mucopus which, in the majority of instances, will not be noticed by the patient as anything more than ordinary nasal discharge.

The whole subject of nasal suppuration demands more consideration from the general practitioner than it has yet had, and in view of the fact that nearly every winter epidemics of grip bring about attacks of acute sinusitis, many of which can be prevented from becoming chronic under proper methods of treatment, it becomes the duty of members of societies such as this, by their written and spoken word, to educate the average practitioner into the need of proper nasal treatment for these conditions in order that they may be recognized early and cases of acute sinus inflammation prevented from becoming chronic.

THE LARYNX IN TYPHOID FEVER.[1]

By Chevalier Jackson, M.D.,
of Pittsburg, Pa.

The vital importance of this subject is better stated by and comes with better grace from a practitioner of internal medicine. Dr. John W. Boyce wrote to the writer as follows:

"The question of great interest is whether it is possible for these affections to cause a fatal termination without such symptoms as shall plainly indicate the nature of the occurrence.

"Theoretically, it seems impossible that a man should choke to death without the conspicuous symptoms of asphyxia, particularly cyanosis; but observation has convinced me that it is only too true; that these cases may sink away as peacefully as a baby goes to sleep, and that one may not in the least suspect what is happening. I am convinced that in times past such has been the case, where I attributed the death to collapse. In the first case of this nature that I ever recognized (though it is scarcely possible it was the first case I ever saw) a fairly acute resident physician had seen the patient, and had no idea that his dyspnœa was in any way an important matter. The case was taken to the operating room for tracheotomy as a precautionary measure, not with the idea that the operation was needed but for fear it might come to be during the night in the absence of a physician. While we were making leisurely preparations for a systematic operation I accidentally noticed that the patient had stopped breathing. Fortunately the surgeon was standing there with knife in hand and was able to stab the patient on the instant. There would not have been time to send to another room for an instrument; as it was, artificial respiration was performed for fully ten minutes after the trachea was opened before the patient could be trusted to do his own breathing. In the last case I saw the patient was rapidly sinking into apnœa, which certainly would soon have been fatal had we not been watching him especially on this identical point; and we had been put on our guard by a slight change of voice, not to hoarseness, but to an aphonia, which I have been accustomed to attribute to relaxation of the vocal cords from extreme weakness and not to lesion. An interesting feature in this case was that the blood pressure as determined by the sphygmomanometer rose when the trachea was opened instead of falling, as we might expect when asphyxia is relieved. Yet there is no doubt whatever that this patient was in desperate need of air before the operation."

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[1] Read at the meeting of the American Laryngological, Rhinological, and Otological Society, Boston, June, 1905.

LITERATURE. Instead of a review of the literature, which space forbids, a bibliography is appended. The statements below differ from those in the literature radically in many points. The writer can only say that the following is a record of clinical observations as carefully made and recorded as limited time would permit.

The great prevalence of typhoid fever of the severest type in Pittsburg at all times afforded good opportunities for study. The following observations were made in the wards of the Western Pennsylvania Hospital.

In weighing these observations it must be remembered that they were all made on hospital cases which on admission are characterized by foul oral sepsis, severe toxæmia, and an aggravated type of disease due to neglect, overexertion during the early stages, unregulated diet, unhygienic surroundings, etc.

Three hundred and sixty cases were watched throughout their illness. Three separate types were observed. Subacute laryngitis, without solution of continuity of the mucosa; ulcerative laryngitis; perichondritis with or without chondrial necrosis.

LARYNGITIS. Subacute catarrhal inflammation of the laryngeal mucosa was present in 227 (63 per cent.) of the 360 cases. It seemed similar to that occurring as an independent affection, save that the surface was at all times freely bathed with secretion, much of which came from the deeper air passages.

That the larynx should participate in the general catarrhal state of the various mucous surfaces, especially the respiratory surfaces, seems natural. Were it not that a few cases develop more serious and possibly fatal lesions the subject might not merit the two years' study here recorded.

ULCERATIVE LARYNGITIS. *Frequency.* The frequency in private practice is unobtainable, but it must be much less frequent than in hospital experience. No reliable records of observations, systematically made on a large scale, are to be found in the literature. The frequency might be supposed to vary in different epidemics, different countries, and in different conditions and environment.

Number examined	360	cases.	
Ulcerative laryngitis in	68	"	(18.9 %)
Started as abscess of larynx in	4	"	(5.9 ")
Required tracheotomy	8	"	(11.8 ")
Perichondritis with necrosis in	6	"	(8.8 ")
" without necrosis in	11	"	(16.2 ")
Abscess from perforation in	2	"	(3.0 ")
Ulcerative trachitis in	9	"	(13.2 ")
" " with perforation and emphysema in	1	"	(1.5 ")
" " with abscess thyroid gland	1	"	(1.5 ")
Pus foci in remote locations in	15	"	(22.0 ")
Associated with acute purulent otitis media	11	"	(16.2 ")
" with leukophlegmasia	6	"	(8.8 ")
Died	4	"	(5.9 ")

BACTERIOLOGY. The typhoid fever bacillus, unaided by other organisms, has been proven to be pyogenic by a number of observers, though it probably is not often active as such without the aid or subsequent activity of pyogenic cocci. It has been found by various observers in abscesses in almost all tissues of the body, and by these observers is regarded as a prime factor. To the writer it seems reasonable to suppose that, excluding one infection (syphilis), the conditions of the soil are more active factors than the seed in any infection. In typhoid fever we have the conditions of soil for the rapid development of pus cocci. In the cases examined the bacillus typhosus was found in the saliva and in the mucosa, but not in the submucosal abscesses. It is not argued from this that it could not have been the prime factor next to conditions of soil. It is more likely, however, that the ever-present pyogenic cocci found a foothold in an abrasion of the mucosa or in an inflammation of the mucosa due to typhoid bacilli lodged from the blood flowing through the mucosal vessels; or a low resistance of cells or juices due to toxæmia; or to a necrosis of the mucosa from anæmia due to embolism or thrombosis, or to an anæmia from any other cause. Or the typhoid bacillus by causing a non-inflammatory tissue necrosis may have prepared the soil for other more active pyogenic bacteria.

The bacillus typhi abdominalis was found in the throat, both on and off the lesions, in secretions and mucosa. It was not found in any greater numbers, however, in cases with throat lesions than in those without. Pus cocci were, however, much more numerous in the cases with throat lesions, after, not before, the ulcerations developed. Of the 4 cases of abscess 2 were found pure streptococcic, 1 pure staphylococcic, and 1 these two mixed.

In one case tracheotomized for stenosis, a subglottic abscess communicating with the necrotic first tracheal ring was found. Pus withdrawn from this abscess with a hypodermic syringe before evacuation proved the pus to contain a pure culture of streptococci. Careful search showed no Eberth's bacilli.

Of the 11 cases of ulceration that also had acute suppurative otitis media it is a striking fact that in every case the same pyogenic organisms were found to preponderate in the laryngeal lesions. Thus of the 2 abscess cases that also had ear suppurations streptococci in pure culture were found in both. In the other 9 the laryngeal ulcerations, being superficial, were necessarily mixed infections, but the preponderating organisms corresponded to those of the ear. Sloughs examined microscopically in 6 cases swarmed with staphylococci, in 4 other cases with streptococci, neither being in pure culture, but mixed with other cocci. Klebs-Loeffler bacilli were found in 1 case only in this gray slough, and were not found in one case where there was an exudation in the trachea.

The following pathogenic organisms were found in the sloughs from the laryngeal ulcers: streptococcus, staphylococcus aureus, staphylococcus albus, pneumococcus, and bacillus diphtheriæ.

LARYNGOSCOPIC APPEARANCES AND CLINICAL PATHOLOGY. Any-one who has watched these cases can make a diagnosis of typhoid fever by the laryngeal appearance and the odor of the breath, which is peculiar, rather offensive, but not putrid. It is as distinctive as that of diphtheria.

Laryngoscopy is not difficult to one accustomed to work, as every laryngologist should be, with the patient in the recumbent position. A self-illuminating laryngoscope is convenient. The secretions should be expectorated and the throat cleared by the patient if possible. The tongue must be handled gently, and care taken that its epithelium does not come away with the towel. There are very few cases in which a good view of the larynx cannot be obtained. Œdema and nothing else may be the picture, the ulcers being hidden.

In one case three blebs were noted, one on the posterior surface of the epiglottis, another on the left arytenoid, and one in the posterior commissure. They contained sanguinolent serum and were followed by ulcers which differed in no way from the ulcers originating as sloughs.

In one case a pseudomembranous exudate lined the larynx and trachea as far as could be seen. This could be wiped away and did not leave a bleeding surface.

Four cases of purpuric spots on the laryngeal mucosa were observed. None of these were followed by ulceration. One of them had submucosal hemorrhage, and all had purpuric spots in the mucosa and skin.

Trachitis without ulceration was noted in 126 cases, though many of these were exceedingly mild. In 2 cases the trachea during life was seen to be ulcerated. One of these ulcers perforated, producing emphysema. In 2 cases tracheal ulcers were seen with the bronchoscope after death.

Of 68 cases of laryngeal ulceration the sites were as given below, a few cases having as many as four ulcers, and some cases having large ulcerations covering more than one location.

Epiglottis	42	Anterior commissure	4
Aryepiglottidean folds	22	Infraglottic region	3
Interarytenoid space	13	Trachea	4
Arytenoids	10	Bifurcation	2
Ventricular bands	7	Bronchi	2
Ventricle	5	Vocal bands	1

It is remarkable that the frequency of involvement should be so nearly in direct relation to the distance from the mouth with the notable exception of the vocal bands, which seemed quite immune. The one case in which they were ulcerated had a specific history and cleared up promptly under specific treatment.

As previously stated the larynx of practically every typhoid fever case was the seat of a catarrhal inflammation, and this did not differ from the acute laryngitis with which we are all familiar. In 5 cases this process seemed to become greatly intensified and the mucosa became diffusely crimson in color, so great was the engorgement. Minute vessels ruptured, apparently from coughing. These hemorrhagic spots later became the site of ulcers in a few cases. In many cases ulceration apparently was not preceded by any diffused increase in the intensity of the laryngitis.

Of the 68 ulcerative cases, 18 had ulcerations of the linear type, 6 were Y-shaped or branched, and 5 were wedge-shaped. Some of these cases also had ulcers of the usual rounded or oval outline.

In ten instances the development of the ulcers was watched from their incipiency. A swelling appeared over an area of mucosa, usually elongated, sometimes linear in shape, and from 2 mm. to 15 mm. in length. At first the swollen area was pale, followed in a few hours by redness. At the next stage there appeared a thin grayish slough, as if the mucosa had been lightly brushed with a solution of argentic nitrate. Through this the deeper structure could be seen to be of a dark purplish color. This faded within a day to an even opaque, dirty gray slough, which separated in a period varying from one to three days, leaving a shallow ulcer, with at first illy defined edges, and a dirty gray bed dotted in a few instances and for a short time with dark-red spots. Later the edges became thickened and the bed covered with a dirty gray exudate. In some instances the ulcer became surrounded by an areola of engorged mucosa, but more often the tissues seemed normal close up to the edge of the ulcer. The depth of the ulcer seemed to be indicated by the thickening of the edges. Soon after the ulceration reached the perichondrium œdema set in. The last two statements are based upon examination from below after tracheotomy. In 5 of the 8 tracheotomized cases the ulcers had thickened edges, and the ulceration had reached the perichondrium. The other 3 tracheotomized cases were abscesses, not ulcers.

In 1 case an abscess of the thyroid gland was opened at the tracheotomy, and the first and second rings of the trachea were found necrosed. The abscess was walled off from the neck tissues but was burrowing under the tracheal mucosa, which was pushed inward, occluding the tracheal lumen almost completely. The patient recovered promptly.

ETIOLOGY. In considering the etiology, first place must be given to the typhoid toxæmia. The blood, tissue, and juice changes are of the most profound character, and this must be borne in mind in considering every other element, as it renders operative otherwise insignificant factors. Slight local trauma, or pathologic change, that in health would be promptly cured by nature, becomes formidable in the absence of resisting power.

It is an accepted fact that a particular type of complications may characterize one epidemic of typhoid fever and a different type another. In Pittsburg we have typhoid fever with us all the time and not only as an epidemic. Yet the tendency one season to preponderance of one type of complications is very noticeable. This has to be considered in weighing etiologic factors. There must be a cause for the preponderance. When the pervading type of complication is purulent and occurs in one ward or institution the natural conclusion is that it is infective and that the source is a previous case. This was particularly noticeable in the purulent complications in the Western Pennsylvania Hospital, which nearly ceased after the elimination of flaws in the technique of disinfection of wards, beds, mattresses, pillows, etc., discovered and righted by Dr. Thomas Arbuthnot. There are several weak points in any argument based on this observation. The same tendency to type is noticed in widely scattered cases in different environments. It is well to note the tendency to laryngeal implication in certain diphtheria epidemics.

One case in 68 (1.47 per cent.) occurred before the twenty-first day. Thus, 98.53 per cent. occurred after the twenty-first day. In 8 cases that died before the twenty-first day no laryngeal lesions were found. In 8 cases that died after the twenty-first day ulcerations existed in the larynges of 2. These 16 examinations were made on the cadaver with a self-illuminating laryngoscope on cases in which autopsy was not permitted, and are not included in the 360 cases systematically examined.

These facts point to the existence of an etiologic factor after the third week that does not exist prior to that time.

What are the characteristics of the third week and the following weeks of typhoid fever as compared with the earlier period? Sensitiveness to cold, a rapid, often sudden emaciation, lowest resistance of the tissues and juices, thrombotic phenomena, glandular disturbance, pneumonia, and bronchopneumonia beginning inflammatory and purulent troubles, separation of the sloughs from the enteric lesions. The worst of the high temperature is over, but its damages remain.

The etiologic possibilities may be classed as follows:

PREDISPOSING FACTORS. 1. Dorsal decubitus.

2. Lessened cough reflex.

3. Lessened frequency of deglutition.

4. Lowered resistance and weakened phagocytosis or its equivalent, due to toxæmia, etc.

5. Increased virulence of organisms proliferating in the air passages.

6. Intensity of the general infection: (*a*) pyrexia and (*b*) toxæmia.

7. Poor blood supply to larynx, predisposing to necrosis.

8. Pretyphoid disease: (*a*) local and (*b*) general (syphilis).

9. Draughts of air, especially when patient is perspiring.

10. Hygienically bad surroundings. Crowded wards.

11. Hydrotherapy: (*a*) sponging, (*b*) tubbing, (*c*) wet-pack, (*d*) ice-pack, and (*e*) ice-bag on head.

12. Drugs.

13. Age.

14. Sex.

15. Nationality.

16. Occupation.

EXCITING FACTORS. 17. Infection *via* (*a*) blood. Typhoid fever bacilli or pus cocci in infected emboli or floating free; (*b*) abrasions from coughing trauma or screatus.

18. Thrombosis: (*a*) aseptic clot (stasis), (*b*) infective emboli (classed above), (*c*) endophlebitis.

19. Oral sepsis.

1. That dorsal decubitus can favor thrombosis by hypostasis the writer does not believe. Anyone who will look along a ward will see that the larynx of nearly every patient is higher than any other portion of his body except his face, varying slightly accordingly as pillows may be arranged. But it seems probable that the sagging laryngeal cartilages may, so to speak, pinch the mucosa between themselves and the vertebra in the supine patient.

2. Lessened cough reflex by favoring incubation of organisms and the development of more virulent cultures in the accumulated secretions is a factor.

3. Lessened deglutition may be a factor in a similar way.

4. Lowered resistance.

The lessened resistance of the tissues in typhoid fever was demonstrated by Brieger and Ehrlich. Malignant œdema followed the injection of a solution of musk in typhoid fever patients, but was harmless in others.

Liebermeister years ago wrote: "If one studies closely the manifold complications and sequelæ of typhoid fever, in the living and at autopsies, he will receive the impression that in severe cases the resistance is reduced to the minimum in all organs without exception, and that there is an extraordinary tendency to the destruction of the tissue." This expresses the clinical aspect of the predisposing factors. It is a condition probably brought about by the action of the toxins on the tissue cells, and is evidenced by the rapid formation (often in twenty-four hours) of large abscesses in various parts of the body. That the vulnerability of the respiratory mucosa should be similarly increased is a reasonable conclusion, while it is, more than other tissue, exposed to pyogenic cocci.

During the time these observations were in progress careful watch failed to find laryngeal ulceration in any other than typhoid fever cases with one notable exception, a case of pulmonary tuberculosis, which was sent in through error and could not be got out. Laryngeal ulceration developed in this larynx while in a room just off the typhoid fever ward. The ulcers were not tuberculous and could not be distinguished from those occurring in typhoid fever, except that the typhoid fever breath was lacking.

5. The possibility of increased virulence was not investigated, but it is deserving of research.

6. As stated, the chief etiologic factor is the typhoid fever. This renders the mucosa vulnerable and thus permits activity of other factors which otherwise would fail. Unlike the complications of the exanthemata, ulcerative laryngitis complicating typhoid fever bears as to the likelihood of its occurrence, its course, and its termination, a close relation to the severity of the primary disease. Under the head of frequency it was mentioned that inquiry among physicians in general practice developed the fact that those practitioners in the poorer districts occasionally saw cases of laryngeal complications, while many of those practising among the wealthy class never saw even one case. The reason for severe cases in the poorer districts seems to be the continuance at labor in the earlier stages (some men working with a temperature of 103°), and at the same time eating a general diet, often forcing food down in spite of anorexia, in the ignorant hope of keeping up the strength. Many laborers make an untreated walking case out of the early stage conditions that would put the wealthier man in bed with bi-daily visits of the physician and the constant attention of two trained nurses to enforce dietary and other restrictions and a proper regime. Another reason may be in the filthy habits and unhygienic surroundings of the poor. The importance of the bathtub in prophylaxis is unquestioned.

The writer's records show that out of 68 cases of ulcerative laryngitis 61 (89.7 per cent.) occurred in cases in which the temperature exceeded 104.2° every day for a week or more, and in 23 (33.8 per cent.) the temperature reached 105°. All other symptoms in a fair proportion of the cases kept pace with the temperature, as ndications of the severity of the case. No case of laryngitis occurred in any case in which the temperature did not exceed 103°. In considering the temperature it must be remembered that except in a few cases not thought to require it the patient was sponged every time the temperature rose above 103°. Wet-packs, and in some cases ice-packs, were used if sponging failed to bring the temperature down.

Von Ziemssen thought the pyrexia was the most important factor in lowering resistance of tissues and advocated antipyretic treatment as the best mode of preventing laryngeal complications. As to the causative influence of pyrexia, this was disproven by the writer's records. Of 10 cases allowed (for other reasons) to go without sponging there were 2 (20 per cent.) developed laryngeal ulcers. This is about the same as the record of all cases, 68 out of 360 (18.8 per cent.). So that it would seem that it is the toxæmia that lowers the resisting power, and that the pyrexia is but an index of the toxæmia.

As to the effect of antipyretics other than hydrotherapy no records were obtainable, as none other is used by the staff at this hospital.

7. The etiologic possibilities of poor blood supply must be considered in the light of pathologic stasis.

8. Local diseases of the upper air passages were faithfully recorded but seemed to have no bearing except on three points. Of 68 ulcerative laryngeal cases, 10 (14.7 per cent.) were mouth breathers, 13 (19.1 per cent.) had chronic laryngitis, and 66 (97 per cent.) had a foul oral sepsis, with foully carious teeth; and of the 6 cases of perichondritis, 3 (50 per cent.) were hucksters, with a chronic laryngitis when admitted.

Several observations seem to point to syphilis being a factor in the etiology of ulceration. Histories, proverbially unreliable, were of even less than usual aid, because of toxæmic stupor, lack of an interpreter, which often rendered it impossible to separate syphilis and gonorrhœa.

Giving the record, however, for what it may be worth, specific history was: positive in 8 cases (11.7 per cent.), negative in 41 cases (60.3 per cent.), unobtainable in 19 cases (27.9 per cent.).

Specific treatment was of prompt and decided benefit in nearly all cases. But mercuric preparations and the iodides are good excretory stimulants and would render good aid in whipping up the overworked excretory apparatus and thus help to rid the system of typhoid fever toxins. This weakens the test value of therapeutics. In general syphilis favors suppurative conditions; so does typhoid fever. The concurrence of the two probably intensifies the tendency.

9, 10, 11, and 12. A chart of the beds showed that one bed located close to a door was occupied by seven successive cases of ulcerative laryngitis. No other one bed had more than two to its credit. This door opened on to a short, draughty corridor which led to a room filled with scrubbing sinks, sewer connections, and water-closets. This corridor, on account of defective plumbing (since refitted), was always overventilated, which caused a draught to strike the bed in question. The proportionate share of the defective plumbing, the draughts of air on the sweating patient, and the defective sterilization of bedding which existed there the writer is unable to state, for all have since been corrected; and while acknowledging a weakness for experimental research, he could not, of course, endorse the reinstatement of these horrors one at a time to determine the share of each. He prefers to put them all down as etiologic factors of the first importance.

Overcrowding of wards is certainly an etiologic factor.

In regard to the effect of a draught of air he must acknowledge his inability to demonstrate wherein a draught of air on a perspiring skin differs from a cold wet-pack. If exposure to draughts of air under certain conditions may cause laryngitis in the well, why may they not so act in the typhoid fever patient?

It is Dr. John W. Boyce's practice to omit sponging in certain cases, no matter what the temperature.

He also does not advise the ice-pack. He stimulates and feeds sparingly. Other members of the staff feed and stimulate more freely, the stimulants being chiefly strychnine and whiskey, and some of them use ice-packs freely. Various drugs such as acetozone and guaiacol were tried extensively by Dr. Lawrence Litchfield. Some patients liked an ice-bag on their heads and were allowed to have it constantly, while others objected to it and it was not used.

Detailed tables would be too voluminous, but the result of observations of all these differences in treatment and management led to the conclusion that hydrotherapy, including ice-packs and head ice-bags (tubbing not being used); drugs, including stimulants, and foods and feeding are absolutely negative as etiologic factors in ulcerative laryngitis, with the single exception of the ice-pack, which had some evidence against it.

13. The age of the patients with ulcerated larynges ranged from sixteen to fifty-five years, but 71 per cent. of them were between the ages of twenty and thirty years.

14. In the female wards 3 cases of ulcerative laryngitis developed in 60 cases of typhoid fever. In the average the typhoid fever cases in the female wards were of a less degree of severity than those in the male wards, probably on account of the earlier taking the bed in the initial stages and other recognized causes. These 3 were very severe cases. As this simply brings us to the severity of the general infection, it cannot be said that sex is a factor.

15. The bearing of nativity on the etiology is interesting. Of 68 cases of ulcerative laryngitis the following were the nativities:

Arabia	1	Russia (Northern)	2
Austro-Hungary	25	Slavonia	...
Ireland	2	Syria	1
Italy	21	United States (negroes)	3
Poland	4	" " (whites)	2
Russia (Southern)	7		

The preponderance of the natives of Southern Europe is interesting, though environment and the fact that they now constitute our laboring class must be considered.

16. *Occupation.* It is worth noting that 3 cases of perichondritis occurred in "hucksters" who call their wares loudly from a wagon in the streets. They had severe chronic laryngitis with hyperplasia and dilated mucosal capillaries on admission.

17. *Infection.* There is much argument in the literature to show that the laryngeal lesions are true typhoid fever glandular involvements similar to the intestinal lesions. Local infection of the lymphoid tissue of the larynx by the bacillus typhi abdominalis is of course a-possibility, but if so, in view of the cases here recorded, it must occur late when immunity is being developed.

Of the 68 cases but 1 (1.47 per cent.) occurred in the first week. It was a diffuse inflammation starting in the lymphoid tissue of the larynx. This case was probably a true typhoid fever inflammation of the lymphoid tissue of the larynx, though the local bacteriologic examination was negative. This was, however, the only case that could be so classed. The other 67 cases all occurred so late in the disease that they could not be considered true typhoid fever lesions in the sense that the lesions of Peyer's patches are. Enteric reinfections occur at this late period and the laryngeal lesions might be regarded in this light, though such is not the writer's opinion. The outline too of these ulcers, as well as the locations, negative the assumption in the literature that the lesions are specific typhoid fever ulcerations, starting in glandular tissue. Of 68 cases, 42 (61.8 per cent.) had ulcerations on or near the free border of the epiglottis where adenoid tissue does not exist.

Local pyogenic invasion assisted by trauma seemed to account for some of the cases observed. Two chief causes of trauma are the "pinching" mentioned under dorsal decubitus and cough. Coughing induced by bronchial conditions was present in nearly all severe typhoid fever cases. This cough is, of course, an irritant to the larynx favoring abrasion as well as inflammation, though of course it is highly necessary to rid the air passages of infective secretions. A longer sojourn than in the normally active state of the cough reflex probably favors the development of a virulent culture.

Two very clear cases of local pyogenic invasion occurred. In both a stream of pus from an acute suppurative otitis media was draining into the laryngopharynx from the Eustachian tube mouth. The ulcers were extrinsic (confined to the epiglottic and posterior cricoid mucosæ), and disappeared promptly after circumcision of the membrana tympani with adequate "wick" drainage through the external auditory meatus.

18. The period and the suddenness of the onset, together with the laryngoscopic appearances as already described, especially the linear wedge-shaped and branched outlines, point to a local thrombosis as one of the chief exciting factors.

As to the etiology of this thrombosis, whether it be primarily an aseptic clot from stasis, or from an endophlebitis, or an endarteritis, or an embolus (aseptic or infective), the writer has only two postmortem findings to offer. Two cases with the linear and branched ulcerations had multiple infarcts in lungs and kidneys. These were the only autopsies. Of course the arteries of the larynx are not "terminal arteries," and thus are not subject to infarct exactly as those of the lungs and kidneys are, but arterial occlusion would by ischemia favor limited necrosis and bacterial invasion.

An embolic etiology would require an original focus in the lungs or left heart or in the periphery proximal to the laryngeal arteries. It is to be regretted that the occurrence of endocardial and other possible lesions bearing on embolic etiology were not recorded.

Several arguments against the thrombotic pathology might be advanced. The number of cases of ulcerative laryngitis associated with leukophlegmasia was small (6). Yet each of these had ulcerations of the linear type. It might be argued that femoral phlebitis and thrombosis is a common complication in private practice, while laryngeal ulceration is rare. And certainly the writer must acknowledge that he has seen but one case of laryngeal ulceration outside of a hospital. But it must be remembered that he has never had an opportunity of systematically observing the throats of typhoid fever cases in private practice. It may be frequent yet, as pointed out by Dr. Boyce, unrecognized, even though it actually be the cause of a fatal termination.

19. Oral sepsis is closely related to infection. Its part seems so self-evident as to require no proof.

Glossitis was present in 2 cases and in 1 case through extension by continuity was the cause of perichondritis, chondrial necrosis, and abscess with subsequent ulceration.

SYMPTOMATOLOGY. The writer considers it his duty to state as a preliminary that all the classic symptoms of laryngeal trouble may be absent, or so slight as to escape notice, being masked by the general malady. Categorically the local symptoms that may be noted are: pain and tenderness, stridor, dyspnœa, dysphagia, odynphagia, dysphonia, odynphonia, hoarseness, croupy cough, and external swelling over the thyroid cartilage. Expectoration is masked by bronchial expectoration.

The general symptoms occasionally seen are recrudescence of pyrexia and extreme prostration with symptoms of approaching death by toxæmia and exhaustion. The pyrexia, as in all post-typhoid fever conditions, usually partakes of the type peculiar to the general malady. In a few of the writer's observations the laryngeal complication only reached the stenotic stage so long after the general toxæmia that the dyspnœa was of the usual active type. But in the feeble state of the profoundly toxæmic typhoid fever patient it was not the symptom we see elsewhere. In 4 of the patients saved from asphyxia by tracheotomy there were none of the symptoms we are accustomed to look for—no sitting erect, gasping, stridor, struggling, violent action of the accessory respiratory muscles, cyanosis, etc. In 3 others slight cyanosis was present and some effort was being put forth, while only in a few cases was the patient making a typical fight for air.

Dyspnœa when present may be due to œdema, occlusion of the respiratory lumen by a projecting abscess, occlusion of the glottis by a fragment of necrotic or sloughing tissue, collapse of the cartilaginous box from necrosis, fixation of, or abductor paralysis of, the cords.

Hoarseness is the most frequent symptom; aphonia next.

The usual cough of typhoid fever was present in 50 (5.9 per cent.) of the 68 cases, but in only 36 of the 50 could it be said to be changed in character after the onset of laryngeal lesions. In a few of these it became quite hoarse and croupy.

The onset of symptoms, averaging all cases that had symptoms, was the thirty-first day. The onset of ulceration in all cases averaged the twenty-second day.

DIAGNOSIS. Diagnosis is easy if the larynx be examined regularly. If reliance be placed on a watch of the symptoms not more than 1 case in 10 will be diagnosticated, and, as pointed out by Dr. Boyce, a certain number will die without the diagnosis ever being made, even in some of the instances in which the laryngeal condition is the real cause of death.

PROGNOSIS. As to life, the prognosis is good if considered apart from the primary general infection. If watched by one skilled in the use of the laryngoscope there is little danger if the suggestions given under treatment be followed. Practically, however, it is not possible to separate the prognosis, and when it is considered that the laryngeal lesion appears in practically always the profoundly toxæmic cases the prognosis as to life must be said to rest altogether on the patient's general condition. As a matter of statistics no case of the 68 died of either the laryngeal lesions or the tracheotomy done for their relief. Only one tracheotomized case died and that was of toxæmia. Of the 68 cases, 4 cases (5.8 per cent.) died; 1 of relapse with intestinal perforation, 2 of toxæmia, 1 of pneumonia. The mortality (5.8 per cent.) seems low, considering that all were severe cases, but it must be remembered that the laryngeal ulceration only appeared after the patient was convalescent and had run the gauntlet of his greatest dangers. How many early fatal typhoid-fever cases would have had laryngeal lesions had they lived of course is beyond human knowledge. In 3 cases ulceration perforated into the soft tissues of the neck, but in no case was there a cellulitis or violent inflammation of the extralaryngeal tissues.

No case need die from the throat complication. The greatest danger to life is from failing to recognize the existence of a laryngeal lesion. Tracheotomy is wrongly regarded as an operation dangerous to life. It has been credited with too many deaths that were justly attributable to the condition calling for it. Of course, as it is the profoundly poisoned cases that get ulcerative laryngeal lesions the prognosis as to life must be guarded, but not because of laryngeal dangers.

As to Voice. In 45 out of the 68 cases (66.2 per cent.) the ulceration healed without alteration in voice or structure, no visible scar remaining. Permanent deformity, without loss of voice and without stenosis, resulted in 8 cases. Stenosis requiring surgical relief to enable the abandonment of the tracheal cannula resulted in 5 cases, all of which recovered good, useful voices of rough quality and uncertain pitch. Cicatricial stenosis can always be avoided if the suggestions under treatment be followed. If it should occur from neglect of early tracheotomy it can be cured with good voice by thyrotomy and prolonged intubation. As to paralysis and fixation, they cease to affect the voice ultimately though their stenotic effects cease only by operative measures.

TREATMENT. In those cases of ulcerative laryngitis that are primary specific typhoid fever lesions, and also those that are of hæmatogenous origin, whether by embolism, thrombosis, or blood-borne pyogenic or specific typhoid fever organisms, prophylaxis is hopeless. But even in these cases the writer does not hesitate to say that if closely watched and judiciously treated no ulcerative case need go on to chondrial necrosis and laryngeal stenotic deformity. These occur in the lately discovered cases and in those cases in which a foul state of oral sepsis, an untreated mucosa, and a long postponed tracheotomy favor a great destruction of tissue.

Prophylaxis then must seek to hold in check the mixed infections that run riot.

The Room. The air should be kept as free from pus organisms as possible, and all cold draughts of air should be avoided, especially if the patient is perspiring. Mattresses and pillows, the entire room in fact, should be sterilized by thorough fumigation. In hospital wards overcrowding must be guarded against.

The Patient. Careful and persistent attention to oral antisepsis (asepsis being impossible) is the first consideration. A dentist's aid should be sought, a daily brushing of the teeth with a soft brush if the gums be tender, followed by swabbing with a one-half of 1 per cent. carbolic acid solution, with borax, sodium bicarbonate, menthol, alcohol, and camphor-water is the most agreeable and refreshing as well as cleansing agent. The patient should be encouraged to clear his throat frequently. All food and water should be sterile. From a laryngologist's viewpoint the ice-pack should not be used unless deemed necessary to prevent a fatal degree of hyperpyrexia.

The writer does not commend the spraying of the nose except where there is a tendency to accumulation of dried or crusted secretions. Usually the natural posterior drainage of the nose, favored by dorsal decubitus, is sufficient to keep the nose clean.

In all the prophylactic measures one thing must be clearly borne in mind, namely, anything that excites or annoys the patient is a detriment to his general condition, so that we must be very sure that what we do is of unquestionable value. The regular toilet of the mouth is agreeable to the patient and is certainly a good thing from the laryngologist's as well as the attending physician's viewpoint, and need not annoy the patient.

In the luetic cases specific treatment yielded prompt results. In other cases considered able to stand it the same treatment yielded good results, probably by acting as an excretory stimulant assisting in the elimination of toxins.

Inhalation of benzoin from hot water is useful. The room may be steamed with water kept boiling to which tincture benzoin compound is added.

In acute œdematous stenosis adrenalin solution 1: 1000 (or any of the similar preparations) as a spray is useful to gain time and may tide over a danger point, but it may become a danger if it lead to too long a postponement of a tracheotomy.

Intubation is useless in œdema, worse than useless in abductor paralysis. If used for the latter, glottic closure will terminate the case promptly should the tube be coughed up.

Of the 5 cases of cicatricial stenosis 3 have been cured by thyrotomy, followed by intubation. They have good though hoarse voices, the tracheotomy wounds are healed and ample laryngeal lumen has been obtained. The method of treatment was given in a paper by Dr. Ewing W. Day and myself, read before the Middle Section of this Society in 1904. One, the last, is still under treatment, and 1 was cured by prolonged intubation with large special tubes.

If closely watched and tracheotomized early, chondrial necrosis with its attendant laryngeal deformity, stenosis, alteration of voice, can all be prevented. All lesions disappear as if by magic after an early tracheotomy. Inability to abandon the tracheal cannula indicates too long a postponement of tracheotomy.

Direct medication to the interior of the larynx is easily accomplished through the tracheotomy wound, though these cases seem to recover promptly without medication after the trachea is opened.

Abscesses may be incised endolaryngeally if desired, but preparations for tracheotomy should be first made, as it may become urgently needed. Personally, the writer would advise tracheotomy with subsequent incision of the abscess through the wound.

With regard to tracheotomy it is practically harmless if Schleich infiltration anæsthesia be used, if it be bloodless, if the patient be in the Trendelenburg position for the operation, and the day after. If tracheotomy be attempted with a general anæsthetic it will usually end up with respiratory cessation and a stabbing operation. Then we have added to the shock of a general anæsthetic the loss of blood, lack of time for hæmostasis. A few days ago Dr. John W. Boyce locally anæsthetized with salt solution a boy of fourteen years who had been terrified by being told (prior to admission) that he would die on the operating table. Yet he made no resistance and acknowledged no pain when the writer did the tracheotomy.

Toxæmia and also carbonic acid narcosis lessen the need for a general anæsthetic in typhoid fever cases, though no case should be allowed to go on to carbonic acid narcosis. After the respiratory centres are profoundly poisoned recovery may be impossible.

The writer here dwells at some length, as he feels strongly on this subject. The astonishing records appear in the literature of three different laryngeal typhoid fever cases which were considered too feeble to withstand a tracheotomy, yet they lived more than two days after it was decided that they had not sufficient vitality to withstand the operation. From their histories they apparently died for want of a tracheotomy.

PERICHONDRITIS AND CHONDRITIS. Perichondritis and chondritis are so intimately associated that clinically they cannot be separated.

The poor blood supply of laryngeal cartilages, lessened by the feeble circulation induced by toxæmia, and still further diminished by stasis, is undoubtedly one of the most important factors in causing necrosis.

Of the 17 cases of perichondritis, 4 started as abscesses and 13 occurred from deepening of superficial ulcerations.

The cartilages involved in the order of frequency were the arytenoids, the cricoid, the thyroid, and the annular tracheal cartilages. As to the involvement of the cartilages of Santorini and of Wrisberg it was impossible to determine clinically.

The epiglottis, while the site most frequently involved in ulceration, was in no case the seat of chondrial necrosis, probably because of its better blood supply.

The writer wishes he could demonstrate pathologically that there is a purulent diathesis. Every clinician sees and knows a type of patient in which every inflammation runs to pus. Many of these cases of abscess and necrosis seemed to be of this type.

Next to this as a cause of chondrial necrosis the writer believes that lues is a fundamental element, not so much as an active lesion as a predisposing factor.

Resume. From laryngoscopic observations of 360 typhoid fever cases the writer concludes that:

1. Serious and fatal lesions of the larynx are much more frequent than is realized.
2. Death may occur from laryngeal stenosis without even the existence of a laryngeal lesion being suspected in the absence of laryngoscopy. If pain and hoarseness be depended upon a diagnosis will seldom be made. Pain is often masked by toxæmia. Cyanosis and dyspnœa are rare, apnœa is common.
3. Unlike the complications of the exanthemata, ulcerative laryngitis complicating typhoid fever bears as to the likelihood of its occurrence, its course, and its termination, a close relation to the severity of the primary disease.
4. The severity of the laryngeal lesion is in direct proportion to the toxæmia, pyrexia not being in itself a factor, but only an index of the toxæmia.
5. Thrombosis of laryngeal vessels in the mucosa or deeper is probably the most frequent local initial lesion.
6. Mixed pyogenic infections are the rule. Laryngeal lesions due to the bacillus typhi abdominalis are exceedingly rare.
7. Prognosis as to life is good if considered apart from the general malady. Not only the life but the laryngeal vocal and respiratory functions will be saved if a tracheotomy be done early. Death from laryngeal lesion means a death for want of an early tracheotomy.
8. Prophylaxis consists in good ventilation, without draughts, sterile bedding, oral antisepsis, sterile food, and water.
9. *Treatment.* Potassium iodide, hydrargyrum biniodid, benzoine inhalations, and oral antisepsis are the best remedies. Early tracheotomy under local (Schleich solution) anæsthesia will cure almost every case.

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In conclusion, the writer urges the laryngoscopic watch of the typhoid fever convalescent or rather defervescent.

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ANGIONEUROTIC ŒDEMA INVOLVING THE UPPER RESPIRATORY TRACT.*

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Angioneurotic œdema, as defined by Collins,[1] is a "vasomotor neurosis or an angioneurosis, characterized by the appearance of circumscribed swellings on various portions of the surface of the body and the mucous membranes, by preference the face, throat, and extremities, without apparent cause or premonition, and non-inflammatory in character."

As it affects the skin, the disease was first described by Quincke as "An Acute Circumscribed Œdema," and from his first description the disease was early called "Quincke's disease." Shortly after Strübing[2] described it as affecting the throat, calling it an "angioneurotic œdema of the throat," regarding it as a vasomotor neurosis, developing rapidly, without fever, after exposure to cold or some traumatism.

The disease is frequently described in books on diseases of the skin under the heading of urticaria. Crocker regards it simply as one of its varieties, giant urticaria. Stelwagon treats of it under a heading of its own, recognizing its similarity in many cases to urticaria, but in other cases says it is entirely or sufficiently independent

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